UNIVERSITY OF TORONTO
MEDICAL JOURNAL

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APRIL 1958

VOLUME XXXV NUMBER 6

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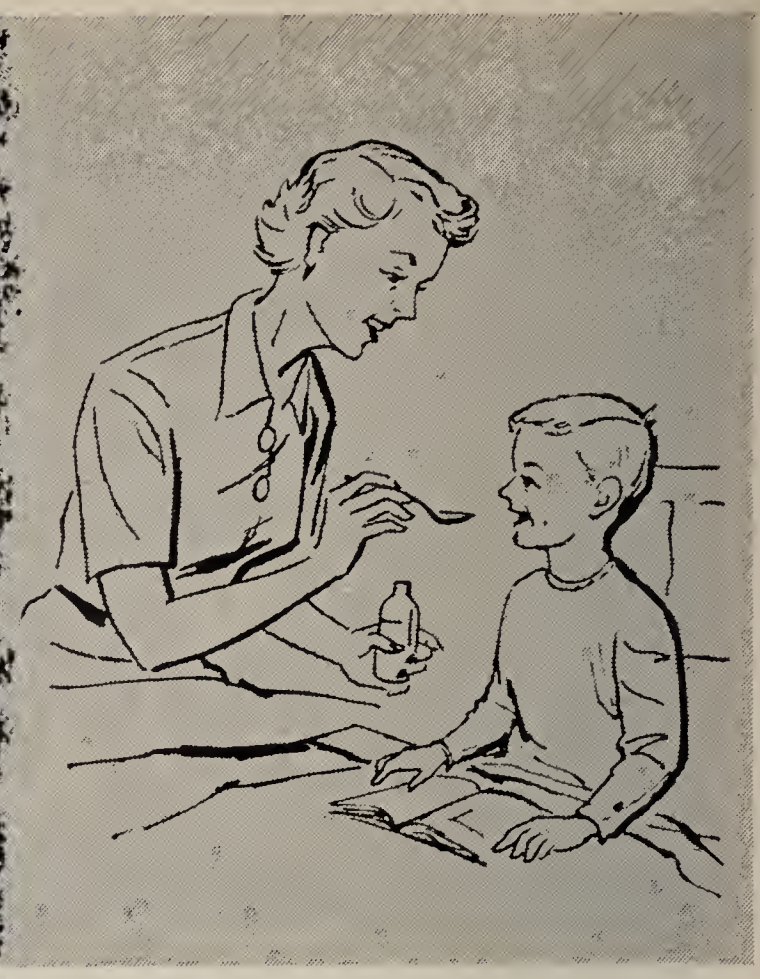

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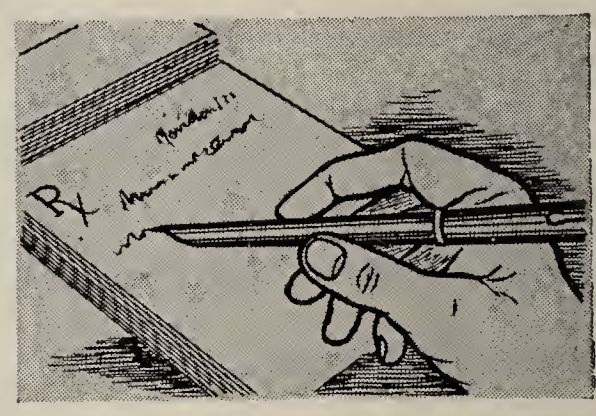

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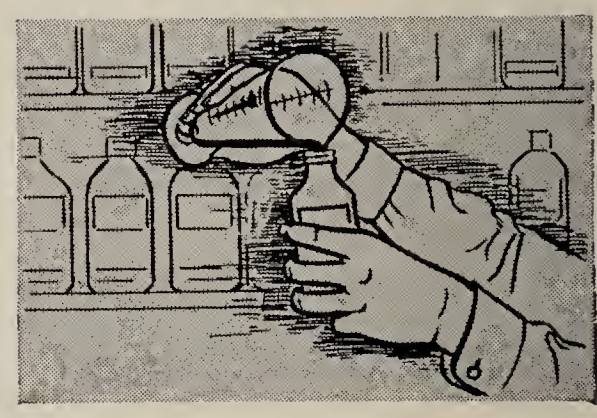

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UNIVERSITY OF TORONTO MEDICAL JOURNAL

ESTABLISHED DECEMBER 1923
AN UNDERGRADUATE PUBLICATION

VOLUME XXXV NUMBER 6

TORONTO, APRIL, 1958

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This Journal is published six times a year from November to April, by the Medical Society of the University of Toronto, to serve primarily as a medium for student expression.

Subscription rates: $3.50 for one volume of six consecutive issues. Single copies 65¢.

Manuscripts including references and footnotes should be typewritten double spaced on one side of the paper. References should be kept to a minimum, and should be cited numerically in the order in which they appear in the text.

Manuscripts in excess of 3,000 words will be considered *unsatisfactory*.

Illustrations are included by arrangement with the Editor. Drawings should be done on white paper in black ink.

Final copy date is the first day of *two months* prior to publication, e.g., September 1 for the November issue.

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Correspondence should be addressed to Room 114, Medical Building, University of Toronto, Toronto 5, Ontario.

Authorized as Second Class Mail, Post Office Department, Ottawa.

EDITORIAL

Finale

Volume XXXV is complete. It began to take form in April 1957 when dozens of topics were presented to prospective authors in the Class of 1958. Now after a year of soliciting, writing, reading, correcting, typing, proof-reading, and publishing, this volume comes to a close.

Probably the only unique feature of this year's Journal was its punctuality. Classes expected, and received, a new issue on virtually the first day of the publishing months. This, we feel, was a triumph, for many previous volumes have been ludicrous in their tardiness. Delivery of the Journal on time can, and should, be achieved. The most gratifying comments received were with respect to this.

Hoping to insert some variety into the pages of print, we presented photographs illustrating medical conditions, signs, and apparatus. These were well received although some people felt that the problems were often too difficult. Unfortunately, because of the cost involved, this feature had to be eliminated from the final two issues. We are grateful to Mr. R. C. Bull, 5T8 and Mr. B. M. Stewart, 5T8 for obtaining and commenting upon these pictures.

To the Juniors

The perennial problem of the Journal offering nothing to the premedical and first medical years again came up. The main argument is based on the fact that every student in this faculty pays a small amount for the Journal. This comes out of the incidental part of his academic fee. The junior students feel that if they are paying for the Journal, they should be able to read it. These students perhaps do not realize that they are also subject to "hidden taxes" with respect to the university health service, university athletics, and the S.A.C., including the Varsity. I wonder how many students go through medicine without utilizing the health service, and although they may be able to read the Varsity, it is unlikely that many of them have their education furthered by it. It should be pointed out to the dissenters that this publication is a *medical* journal, and that it represents the undergraduate body of this faculty on library shelves of medical schools and medical centres on a national and international scale. It is probably not common knowledge that requests for reprints come to contributors to this Journal from as far away as Montreal, Alberta, the United States, and even medical centres in Europe. This, we feel, is very gratifying and encouraging. This Journal is respected by other medical schools and other medical centres; it is about time that a similar respect was paid by the junior undergraduates of this medical school.

Acknowledgement

I am most grateful to the many persons who have made this volume possible, first of all, to the writers whose individual efforts to produce papers of good quality are appreciated. The Journal staff has been most co-operative. Mr. P. B.

Dent, 6T0 has handled the advertising with such initiative and organization as to make our financial situation much less precarious and to re-establish good relations with the advertisers. Mr. D. J. Brand, 6T1, spending his first year with the Journal, took over the circulation—a situation characterized by chaos. His efforts to keep the graduate subscriptions up-to-date and re-organize our subscription list have occupied many hours of his time. Mr. H. P. James, 5T9 has conducted the business aspects with responsibility. The abstracts which appeared from time to time are the work of Mr. R. F. Armstrong, 5T9.

Mr. P. C. F. Moore, 5T9 who has been with the Journal since premedical years is now about to receive the helm for volume XXXVI and 1958–1959. I wish him good writers, good material, good advertisers, and above all, good luck.

A C. S.

OTITIS MEDIA

N. D. KERR, 5T8

A THOROUGH knowledge of otitis media is of importance to all practitioners because of the frequency of occurrence and the severity of complications. Although it is a disease largely of childhood, it does carry on, more frequently in its chronic forms, to adulthood. A study of the symptoms and of the current treatment is worthy of our attention and review from time to time.

Definition

Acute otitis media is an acute infective inflammation of the middle ear cleft.

Classification

ACUTE

1. Infancy:
   a. Acute otitis media neonatorum
   b. Acute otitis media of infancy associated with gastroenteritis
2. Later Childhood:
   a. Catarrhal otitis media
   b. Serous otitis media
   c. Suppurative otitis media

CHRONIC (children and adults)

a. Chronic catarrhal otitis media
b. Chronic suppurative otitis media

INFANCY

I. Simple Acute Suppurative Otitis Media Neonatorum

This differs in many ways from the corresponding disease in older children and adults. It can become annoyingly persistent, and at times may become dangerous with dramatic speed.

ETIOLOGY

The higher incidence of O. M. in infants is unquestionably due to their greater susceptibility to infection as a result of their lack of immunity. The nature of the Eustachian tube also provides an explanation for the supposedly greater incidence of acute otitis media in bottle-fed babies. When the infant is fed at the breast, it is normally held in a more or less upright position, but when bottle-fed, the infant may be allowed to lie on its back, thereby allowing milk to find its way into the nasopharynx and so into the tympanum via the Eustachian tube. Also, it should be remembered that bottle milk is more likely to be infected than breast milk; further, the breast-fed baby is more robust than the bottle-fed infant, and therefore more capable of resisting infection.

Teething is often supposed to be a cause of otitis media in infancy. Its occurrence is explained by saying that the pain of teething lowers the general resistance, and renders the child more likely to infection. This is regarded as superstition by authorities now although many poorer-class mothers still regard a purulent otorrhea as a normal physiological accompaniment of teething.

BACTERIOLOGY

a. Predominantly staphylococcus aureus and pneumococcus (including the dreaded Type III).

b. Streptococcus haemolyticus occurs more often with ear infections in later life.

c. B. pyocyaneus may give rise to a troublesome form of otitis characterized by a greenish-yellow discharge of pus with a musty odour.

SYMPTOMS AND SIGNS

Acute O.M. is a common condition in infants. The tympanic membrane appears to be relatively tough, and otorrhea is therefore often a late symptom. The

other two cardinal symptoms, earache and deafness, being subjective, cannot be noticed by the parents, and the infant with otitis media is therefore presented to the doctor as an ill and restless baby. Only rarely is the doctor's attention drawn to the ears by the mother.

The symptoms are almost exclusively those of a general disturbance. The child is clearly in pain, but there may be nothing to suggest that the pain is in the head. In mild cases, the child recovers from this constitutional upset after some hours when a discharge may be noticed in the meatus, although a number of children probably recover without otorrhea—the discharge being evacuated via the Eustachian tube.

If the general illness continues, the infant refuses its food, vomits, continues to be restless, and is obviously in severe pain. The temperature may rise to 105°F., but is intermittent. There is often *no* indication that this febrile illness is due to middle ear disease. It clearly follows that in the presence of unexplained pyrexia, every infant should have its ears examined.

EXAMINATION

The infant is wrapped in a blanket and placed with its head on a nurse's shoulder who steadies it with her hand. The external auditory meatus must be meticulously cleaned of wax, pus and other debris. A small speculum is used, and the auricle is pulled *downward*, not upward and outward as with the adult. A head mirror is more useful than an electric auriscope as the latter requires both hands for its satisfactory manipulation.

COURSE

In acute otitis media the drumhead first becomes dull and apparently thickened. The cone of light disappears and details are gradually lost so it is difficult to identify the short process and handle of the malleus.

As the disease progresses, the drum becomes dull red, first in the region of Shrapnell's membrane (i.e. the pars flaccida of the tympanic membrane) and later in the postero-superior quadrant which may bulge. Finally, the whole tympanic membrane may be deeply red and bulging. If spontaneous perforation does occur the discharge is first serous and blood-stained and later becomes purulent, depending on the stage of the disease.

TREATMENT

General

The treatment is myringotomy carried out under general anaesthesia. Once adequate drainage has been established, the infant's general condition should rapidly improve and penicillin and the sulfonamides can be given to advantage.

Myringotomy may be withheld if the surgeon feels that there is no fluid in the ear. This is often difficult to decide and if in doubt, myringotomy should be done.

If it is thought that pus is in the middle ear, then it is essential to drain the ear before chemotherapy or penicillin is administered.

Local

It is unlikely that any medication given in the meatus will directly affect the progress of middle ear infection. Adequate toilet and maintenance of a healthy state of skin are important. Penicillin and sulfonamide drops are probably useless. It is most important that the meatus should be properly cleaned with full aseptic precautions at least twice daily. The meatus should be mopped out with sterile cotton wool and small applicator under full vision.

Syringing the ear with normal saline solution or with 30% alcohol is of value. Normally the ear will become dry in a

week or so after otorrhea commences. If it persists for four weeks, then a review of treatment is necessary with subsequent drainage of the mastoid.

COMPLICATIONS

1. *Acute Mastoiditis*: There are certain differences between mastoiditis of infancy and of the adult. The type of pain is of no diagnostic value because the infant cannot express any differentiation between pain originating in the antrum and pain due to pus in the tympanum. Fever is common in all infectious diseases of children, and therefore a rise in temperature is less likely to be of clinical importance. Convulsions, hyperpyrexia, sudden chills and vomiting are also of less significance than in the adult as the cerebral centres in infancy are more easily excited by peripheral irritation. Retro-auricular swelling beginning as a puffy oedema and later as a fluctuant swelling usually makes the diagnosis.

Silent mastoiditis due to Type III pneumococcus is an insidious and progressive disease which is afebrile and painless with markedly progressive destruction of bone in the mastoid area. The ear condition may only be discovered in the advent of some intracranial complication. Only tenderness and oedema may be found over the mastoid in a child slightly ill and out of sorts in such a condition.

2. *Intracranial Complications*: Of all the complications, meningitis is by far the most common and occurs more often than in the adult. Extradural abscess, brain abscess and sinus thrombosis also occur but less often than in older children and adults. Lateral sinus thrombosis is in fact extremely rare in infancy. Otic hydrocephalus is a rare complication which occurs most often in children and adolescents, but is occasionally seen in infants.

TREATMENT OF COMPLICATIONS

1. *Mastoiditis*: In infants, the operation is a simple one. The outer cortex of the mastoid is removed exposing the mastoid antrum, and a rubber drain is inserted—the whole operation not taking more than a few minutes.
2. *Intracranial Conditions*: Treatment is essentially the same as in adults.

II. Acute O. M. of Infancy Associated with Gastro-enteritis

Acute non-specific gastro-enteritis of infants is a disease limited to the first year of life. Gastro-enteritis occurring in later childhood does not possess the same characteristics as the disease occurring during the first year. The dominant feature of early gastro-enteritis is severe dehydration which is not seen later on.

It has been shown that gastro-enteritis predisposes to middle ear infection in several ways. The patient's resistance is lowered. If it should vomit when lying on its back, some of the infectious material may pass down the wide Eustachian tube into the middle ear and mastoid antrum. As it makes an effort to swallow the vomitus, the mouth of the Eustachian tube is opened by the tensor palatini, and fluid passes into the middle ear. It has also been shown that B. coli which is the most frequent cause of gastro-enteritis can be isolated and cultured from such middle ear discharge.

SIGNS AND SYMPTOMS

The signs of parenteral acute otitis media differ only in degree of severity from those of acute otitis media neonatorum described above. They include pyrexia, fits of screaming, restlessness and convulsions. Enlargement of the posterior cervical lymph chain is occasionally seen.

"All children with gastro-enteritis should have their ears examined at least once daily and more frequently should

they show a sudden exacerbation of symptoms." The rapidity with which acute O.M. can develop is extraordinary. The drum may deteriorate from normal to spontaneous rupture in a few hours.

TREATMENT

If the drum shows a general or local bulging with the typical opaque dull red appearance or reddening it must be immediately incised. In acute otitis media neonatorum a short anaesthetic is justified, but these children are often not in a satisfactory condition for anaesthesia. Both drums should be opened.

Following myringotomy, a dramatic improvement often occurs. However, the incision often closes rapidly and should the membrane revert to its previous state a recurrence infection may lead to the same chain of events. Evacuation of pus is once more indicated, and this may have to be repeated three or four times.

If the gravity of the child's condition persists in spite of repeated "positive" myringotomies, mastoidectomy must be considered. Indications are:

a. Persistent pyrexia
b. Persistent or increasing diarrhoea
c. Sudden or recurrent dehydration
d. Persistent or profuse otorrhea

Penicillin and other antibiotics give disappointing results but should be employed. It is important to combat the severe dehydration, and blood transfusion saves many lives.

The apparent results of operation are poor—often as high as 50% mortality. These are not true figures, however, for many cases deteriorate so rapidly that they are moribund when they come to operation. It is possible that earlier operation would save a number of lives.

LATER CHILDHOOD

I Catarrhal Otitis Media

This is an altogether milder infection of the middle ear often due to M. catarrhalis, and generally has an indefinite course without marked symptoms.

COURSE

The infection arises from an extension of a nasal, post-nasal or acute upper respiratory infection up the Eustachian tube. Douching the nose, swimming and especially diving are common causes.

The Eustachian tube becomes blocked, and the air throughout the middle ear tract is gradually absorbed so that the air pressure on the outer side of the drumhead causes it to become retracted inwards (but fluid in the middle ear may later cause bulging).

Deafness results but may be intermittent accompanied by earache or fullness in the ear. The patient will complain of headache, increased resonance of his own voice in the head and the occurrence of tinnitus or vertigo.

TREATMENT

This is essentially the same as for a cold in the head—aspirin gr. $\bar{x}$ q4h and a day in bed are usually sufficient. If earache is present, warm glycerin ear drops may be used. If no nasal infection is present, daily eustachian catheterizations may be carried out for one week. The alkali treatment usually disperses the middle ear fluid but if it fails, the drumhead should be incised. Acute catarrh usually clears up in a short time but if neglected may drift on to chronic infection of the middle ear.

II. Serous or Secretory Otitis Media

This is essentially a non-inflammatory condition although it may become inflammatory. It is chacterized by a collection of serous fluid in the middle ear causing some degree of deafness, sometimes slight tinnitus and vertigo, and a feeling of fullness or pain in the ear. This condition is often an extension from the nasal passages proceeding to irrita-

tion of numerous mucous glands in the Eustachian tube with consequent blockage. In some cases the presence of adenoids may be a main factor; in others, allergy may be a cause.

CLINICAL SIGNS

On examination of the tympanic membrane, there is usually at first retraction of the drumhead due to closure of the Eustachian tube. If there is only a small amount of fluid in the middle ear, the appearance of the drumhead will be normal, but as fluid increases a "hair-line" fluid level across the drumhead will be seen and later on the drumhead may bulge.

TREATMENT

Alkali should first be given (e.g. bicarbonate of soda gr. $\overline{xx}$ q2h. for 48 hrs.) to disperse the fluid. Atropine Sulphate gr. 1/100 is useful. Eustachian catheterization should be carried out at once and repeated as often as necessary. Myringotomy may be necessary.

III. Acute Suppurative Otitis Media

This is most common during the first two years of life; in older children the incidence is still greater than in the adult because of the child's lack of immunity and greater liability to infection, especially to coryza and the acute fevers. The presence of excessive and infected lymphoid tissue in the nose and throat is a predisposing factor.

SIGNS AND SYMPTOMS

Following a severe cold in the head, acute otitis media is ushered in by pain in the ear which is persistent, severe and often throbbing. Relief is usually afforded the moment perforation of the drum occurs. In adults the temperature for several days varies from 100–102°F. but in children it rises to 105°F. Chills and even convulsions in very young children may occur. Tinnitus of a roaring, pulsating nature is present. The dizziness is of a tactile nature—the patient stating that when he arises, the foot of the bed goes down. Tenderness over the mastoid is common. The character of the discharge depends on the type of organism present, but in itself is not of any prognostic value.

Hearing is always diminished in suppurative O.M. but clears up with resolution of the process. Tuning fork tests embrace the Weber which lateralizes to the affected side, the Rinne which is negative, and increased bone conduction and reduced hearing for lower tones.

The drum becomes red and inflamed at an early stage with disappearance of landmarks. Perforation usually occurs spontaneously in the antero-inferior or postero-superior quadrant.

Resolution usually occurs in one to three weeks and the perforation remains open for a long time or does not close at all. If it heals over, the landmarks gradually reappear and healing may occur due to calcareous deposits or fibrosis.

TREATMENT

This follows the usual lines with chemotherapy and antibiotic treatment and myringotomy if necessary. Predisposing factors in the nose or throat should later be eliminated.

The patient should be put to bed. If earache is severe, a codeine-aspirin combination will relieve the pain. The instillation of warm phenol-glycerin (5%) drops is a valuable aid as is extra heat. Penicillin or sulphas are valuable. However, too much time should not be lost before providing adequate drainage in the presence of a suppurative process in the middle ear with a bulging ear drum. Here, a general anesthetic for children prior to myringotomy is preferable but for adults, local anesthesia, using equal parts of menthol, phenol and cocaine is adequate.

CHRONIC FORMS

The term, Chronic Otitis Media, embraces different forms of a disease characterized by various symptoms and pathological changes. The problem in a chronic discharging ear is to relieve the patient of the dangers associated with such a disease and to produce, if possible, a dry ear with adequate hearing.

SIGNS OF CHRONIC OTITIS MEDIA

The signs are otorrhea, changes in the appearance of the tympanic membrane and loss of auditory acuity.

Otorrhea: This may be scanty or copious and there is usually a pool in the deep meatus. A copious mucoid non-odorous discharge suggests an infection of the Eustachian tube and mucosa of the membranous portion of the middle ear. A scanty non-odorous discharge suggests an attic perforation without active bone necrosis and is expected to recover with operation. A scanty odorous discharge suggests bone necrosis and cholesteatoma formation. It is unlikely that this form of chronic disease can be cured without operation. A copious purulent discharge suggests acute bone infection and possibly extradural abscess and will probably require an operation.

Site of Perforation: The site of perforation is of great importance. A central perforation has the best prognosis whereas a marginal perforation is more likely to be resistant to conservative measures. A perforation through Shrapnell's membrane offers a poor prognosis even with surgical treatment.

ETIOLOGY

The question may be asked why some ears clear up and others go on to chronicity. Chronic discharging ears usually follow one or more attacks of the acute suppurative types being influenced by local and general factors accounting for continuation of the infection. Measles, scarlet fever and diphtheria often result in chronic middle ear suppuration because the resistance of the patient has already been taxed to the utmost. An atypical anatomic situation with recesses in the developmental structure of the middle ear influences the process. Important also is a persistent nasopharyngeal and Eustachian tube infection. Improper treatment or neglect may be responsible for protracted suppuration.

I. Chronic Catarrhal Otitis Media

This is the end stage of acute catarrhal otitis media.

SIGNS AND SYMPTOMS

The typical case is that of a less violent illness than previously described. Pain is less severe and *not* dramatically relieved by the onset of otorrhea or by myringotomy. It gradually passes off over the course of days or even weeks. Temperature and systemic disturbances are less marked. The otorrhea is less copious and purulent in the early stages, and is frequently no more than a moistening of the meatus with a serous exudate which dries around the orifice of the meatus.

The otorrhea may be continuous with variation in quantity or continuously scanty or intermittent with long intervals when there is no discharge.

TREATMENT

Here, treatment should be directed at the cause. One should try to determine if some pathological process in the nose, nasopharynx or pharynx is responsible. Often infected adenoids are noted or chronic nasopharyngitis, or even a rhinosinusitis may be responsible.

For the ear itself, cleanliness is important. Inflations aid and should be performed at frequent intervals. Heat via short wave therapy is often very satisfactory. General treatment should aim to

control the tinnitus by sedation and to improve the patient's physical state.

II. Chronic Suppurative Otitis Media

CLINICAL PICTURE

The incidence of this disease is much greater than is commonly believed. Chronic suppurative otitis media is a common cause of deafness. These cases have a chronic discharge from the middle ear with a perforation of the drumhead associated sometimes with small and large granulations and sometimes cholesteatomas. Tinnitus is a common complaint.

The potentiality of complicating intracranial involvement in every patient with chronic suppurative otitis media renders it incumbent upon the physician to apply every known conservative method of treatment that holds out hope of cure. Only then should surgery be contemplated.

TREATMENT

a. *Tubo-tympanal Otitis Media*

This is regarded as a "relapsing Eustachian catarrh". The discharge is mucoid rather than purulent and the perforation is central. Destruction of bone does not occur except in the handle of the malleus. Mastoiditis is rare and mastoid surgery is not indicated.

Scrupulous and repeated cleansing of the meatus is important and essential. Antiseptic powders and instillations are secondary adjuncts. Of these, iodine powder and boric alcohol drops are good. Some advocate antibiotic and sulfonamides locally, but these are apt to cause skin sensitivity. Polypi and granulations must be removed by avulsion or silver nitrate application. Nasal sinus disease, infected tonsils or adenoids and any other cause of infection or Eustachian obstruction must be dealt with subsequently.

b. *Attico-antral Otitis Media*

Here the discharge is typically purulent and unless rendered harmless by radical surgery is an ever present potential danger to life. The perforation is marginal and bone destruction is common. The treatment is essentially surgical although it is possible to obtain a cure by conservative means in early cases and in those where the perforation is large and bone destruction is slight. Here, procedures outlined as above may be tried.

However, in many instances conservative treatment will be a waste of time. Radical mastoidectomy is performed in those cases with gross destruction or complications requiring free exposure, and stress is put upon the preservation of function by removing only those structures which must be sacrificed in order to obtain free drainage.

COURSE AND COMPLICATIONS

Chronic Suppurative Otitis Media becomes resolved either spontaneously or under proper treatment. The hearing defect is in relation to the amount of destruction and length of time the suppuration persists. The deafness, despite a large defect in the drum, is not always marked. It is the adhesive process, binding down the ossicles, that leads to profound deafness.

The production of granulations, fistula formation, polyps and cholesteatomata delays healing and predisposes to complications as the result of acute exacerbations induced by the so-called "common cold". The complications are similar to those caused by acute suppuration of the middle ear except that they occur with greater frequency in chronic suppurative disease.

Summary

The two main points to be emphasized in the modern concept of ear treatment and surgery are that one must (1) en-

deavour to produce a dry safe ear and (2) try to get an early return of functional hearing and balance with a minimum of complications and chronic sequelae.

In this paper, the author has attempted to bring these points out by stressing (1) the necessity for early diagnosis; (2) the need for early operation in the acute case; (3) the inadequacy of antibiotics in the face of poor drainage; and (4) the need for subsequent procedures to remove chronic foci of infection in the ear, nose and throat.

Acknowledgement

The author wishes to thank Dr. P. E. Ireland for his helpful criticism in the preparation of this poper.

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INTRACRANIAL SUPPURATION

W. J. REYNOLDS, 5T8

THE incidence of suppuration within the skull has not decreased since the advent of antibiotics, but its prognosis has improved. However, it is still a highly dangerous complication of common infections in the body. This article will deal with the development and clinical features of intracranial suppuration, but treatment will only be outlined.

With the exception of primary bacterial meningitis, suppuration occurs within the skull in five situations: (1) extradural abscess, (2) subdural empyema, (3) suppurative thrombophlebitis, (4) acute localized non-suppurative encephalitis, and (5) brain abscess.

Extradural Abscess

Extradural abscess results from the spread of osteomyelitis in the skull to the underlying extradural space. It occurs as a complication of a compound fracture of the skull, otitis media, or sinusitis. Pus strips the dura off the inner side of the cranium, and a layer of granulation tissue lines the outer side of the dura. The latter is an effective barrier to the spread of the infection, and the abscess may remain localized for a long time.

Diagnosis may be difficult as the abscess is rarely large enough to produce focal neurological signs or a severe rise in intracranial pressure. Local signs of inflammation, that is, swelling, redness and tenderness are present. A high intermittent fever may lead one to suspect the complication. There usually is headache radiating from the mastoid in otitis media or the frontal region in frontal sinusitis; this is valid only if the otitis media or sinusitis is being properly drained, and thus not the cause of the headache. Usually there is marked tenderness of the affected part of the skull; this is an important sign in chronic sinusitis or otitis media. There is a polymorphonuclear leukocytosis, and the cerebrospinal fluid usually shows a slight increase in cells, either polymorphonuclear or mononuclear.

Treatment consists of adequte drainage of the abscess with removal of the infected overlying bone. Suitable antibiotics are administered. The primary infection must also be eradicated.

Subdural Empyema

The source of this condition is usually an acute frontal sinusitis, but an otitis

media, mastoiditis or ethmoid or sphenoid sinusitis may also be the primary infection. Infection is by direct extension, and thus the empyema is associated with a subaponeurotic abscess, osteomyelitis of the skull and an extradural abscess.

PATHOLOGY

The infecting organism is usually staphylococcus, but occasionally pneumococcus. The subdural pus spreads in three directions: (1) under the orbital surface of the frontal lobe and along the Sylvian fissure, (2) along the falx cerebri to overlie the tentorium, and (3) over the convexity of the hemisphere. It may also spread under the falx cerebri to the subdural space about the other hemisphere. A layer of granulation tissue is produced on the inner surface of the dura; there is also a localized subarachnoid exudate underlying the subdural pus, but without generalized leptomeningitis. A brain abscess may or may not be present.

CLINICAL FEATURES

When an empyema complicates frontal sinusitis, there is an exacerbation of frontal pain and tenderness and nasal discharge followed by orbital swelling. Headache becomes generalized, and a clinical picture of a semicomatose, very ill patient with high fever, signs of meningeal irritation and leukocytosis is produced. Seizures are common. There may be focal neurological signs, for example, aphasia in dominant hemisphere lesions, paralysis of contralateral deviation of the eyes, or a field defect. Hemiparesis is usual.

The cerebrospinal fluid shows an increased pressure. Protein is raised often to 100 mg. per 100 ml., but sugar remains normal and the fluid is sterile on culture. A cell count of several hundred is usual with a majority of polymorphonuclear leukocytes.

TREATMENT

The aim of therapy is the evacuation of pus wherever present. Burr-holes are made where pus is expected and the pus is sucked out. A co-existing brain abscess is also evacuated, and the primary infection is drained. Subsidiary to this is correction of improper fluid balance, prevention of the temperature rising too high and correction of anaemia if the pus has been there any length of time. Antibiotics are administered systemically and locally.

A subdural empyema is often difficult to distinguish from an intracranial thrombophlebitis or an acute suppurative encephalitis predominantly involving one hemisphere.

Suppurative Thrombophlebitis

The commonest venous sinuses affected are the lateral in spread of infection from the middle ear, the cavernous in spread from the skin of the face, and the superior longitudinal in spread from the frontal sinus. The thromobophlebitis tends to spread from one sinus to another and from the lateral sinus down the jugular vein. A piece of softened clot may break off and be carried to the lung where the usual result is a pulmonary abscess.

CLINICAL FEATURES

The clinical picture is that of a severe infection with high fever, chills and rigor. In lateral sinus thrombosis there is usually temporal headache; there may be papilloedema, but focal neurological signs with the exception of nystagmus are usually absent. Queckenstedt's test usually produces no rise in cerebrospinal fluid pressure. A cavernous sinus thrombosis is characterized by ipsilateral frontal headache and facial pain followed by proptosis, oedema of the eyelids, chemosis and paresis of the oculomotor nerves. Suppurative thrombosis of

the superior longitudinal sinus usually produces headache with marked papilloedema and some oedema of the scalp over the vertex. Frequently a subdural empyema results.

TREATMENT

Antibiotics are essential in all three situations, and are the only therapy in cavernous sinus thrombosis. Surgical drainage of the lateral sinus may be carried out, but ligation of the internal jugular vein must be done first.

Acute Localized Non-suppurative Encephalitis

A brain abscess has an initial inflammatory response of vascular congestion, oedema and cellular infiltration before there is any localized suppuration. In the days before antibiotics this usually went on to abscess formation if the patient did not die first. In addition to a primary source and general signs of infection, the patient shows evidence of a local disturbance in the brain, and there may be convulsions. An electroencephalogram demonstrates a focus of slow waves.

Treatment is by administration of massive doses of sulphonamides, penicillin and chloromycetin. Close clinical observation is necessary for signs of recovery, such as improvement in level of consciousness, return of temperature to normal and disappearance of focal neurological signs. Before discharge, air studies should be done to ensure that brain abscess has not formed and become quiescent under intensive therapy.

Brain Abscess

Brain abscess comprises the largest percentage of cases of intracranial suppuration. Although there has been a considerable reduction in its mortality from the pre-penicillin rate of 46% to the present rate of about 20%, the latter figure indicates that brain abscess is still a serious condition.

SOURCES OF INFECTION

The commonest source of infection is paranasal sinusitis followed closely by otitis media; in both of these the chronic form is the more common offender. Penetrating wounds of the brain, scalp lacerations and compound fractures of the skull with or without a dural tear constitute other sources. The fact that a fractured skull is not necessary for the intracranial spread of infection (Linnell and Robinson) illustrates the importance of attending to apparently trivial scalp wounds according to established surgical principles. Chest infections such as bronchiectasis, lung abscess or empyema constitute the most lethal source as frequently these give rise to multiple abscesses. It has been established that 5% of deaths from congenital heart disease are due to brain abscess. Various other conditions, for example, boils, carbuncles or valvular heart disease may give rise to brain abscess.

Abscesses of otitic origin are found in the temporal lobe or the cerebellum, spread being a result of (1) purulent thrombosis of the lateral sinus, (2) osteomyelitis of the tympanic roof, or (3) spread along the adventitial spaces of perforating blood vessels. Frontal sinusitis produces brain abscess in the frontal lobe. Haematogenous abscesses may occur in any situation, but are nearly always above the tentorium, and often in the area supplied by the middle cerebral artery.

BACTERIOLOGY AND PATHOLOGY

Any pyogenic organism may produce a brain abscess, the commonest being staphylococcus; however, streptococcus, pneumococcus, members of the B. coli group, Friedlander's bacillus, and even actinomyces or entamoeba histolytica may be responsible. Invasion of the brain leads to focal necrosis and liquefaction accompanied by oedema of the surround-

ing brain tissue to produce suppurative encephalitis. This proceeds to formation of a capsule by fibroblasts derived from proliferating capillaries, and in due time a firm, thick fibrous capsule forms, and may be several millimetres thick. Surrounding this wall in the brain are microglia, fibroblasts and astrocytes.

CLINICAL MANIFESTATIONS

The mode of onset of brain abscess may be acute or insidious. If it follows a compound fracture of the skull, it usually develops soon after the injury, although there may be a latent interval. In the case of otitis media or sinusitis the patient may suddenly develop acute signs of cerebral derangement and increased intracranial pressure, or there may be a prolonged period in which the patient does not appear well being subject to vague headaches, nausea, anorexia and weight loss. Haematogenous emboli may produce a sudden onset of seizures and focal neurological signs followed by pyrexia and increasing intracranial pressure, or the abscess may develop insidiously and often be clinically indistinguishable from an intracranial tumour.

Acute cases usually present with evidence of increasing intracranial pressure and symptoms of infection. There is a deterioration in level of consciousness with falling pulse rate and rising blood pressure. In chronic abscesses evidence of raised intracranial pressure is not a prerequisite to diagnosis; papilloedema may not appear until the disease has become well established. In many chronic cases pyrexia and leukocytosis may be lacking.

An abscess in the frontal lobe may present with aphasia, hemiparesis or conspicuous impairment of memory and attention, but these may not be present, and there may be only a mild reflex change on one side of the body, a little facial weakness, an isolated fit or nocturnal incontinence. A temporal lobe abscess commonly presents with a visual field defect in the opposite homonymous field, or if in the dominant hemisphere there may be a nominal aphasia. A cerebellar abscess often produces a suboccipital headache; there may only be nystagmus, but ataxia and inco-ordination are usually present.

AIDS TO DIAGNOSIS

A lumbar puncture may be helpful in that the cells are usually increased to 10 to 200 per cu. mm. with either polymorphonuclear or lymphocytic cells predominating. Protein is usually elevated to 100 mg. per 100 ml. or more. Culture is negative and sugar and chlorides are usually not decreased. However, a lumbar puncture may be disappointing in that it is far from conclusive especially when the cell count is low. Furthermore, it should not be done if there is evidence of increased intracranial pressure. The electroencephalogram and angiography are helpful in accurate localization of the lesion.

TREATMENT

Perhaps the most important fact to recognize in the management of a brain abscess is that it may become acute at any stage in its development. The principles of management are evacuation of the pus and prevention of extension of the process. In the acute stage in which there is oedema of the surrounding brain with or without an encapsulated abscess, aspiration must be done before tentorial or foramen magnum herniation develop; decompression may be necessary. When there is capsule formation, aspiration is carried out with local instillation of antibiotics and a contrast medium such as thoratrast. Repeated aspiration may be necessary. Bacteriological studies are made and systemic antibiotics are given also. The size of the abscess is followed by X-rays. When the abscess is small and well encapsulated, excision is carried out

if it can be done without increasing the patient's disability or increasing the risk of mortality. Otherwise, it is dealt with by continued aspirations and local instillation of antibiotics until no more pus is formed. Botterell believes that a cerebellar abscess, encapsulated or not, should be excised at once using aspiration only for diagnosis and preliminary instillation of antibiotics, because acute swelling rapidly causes irreversible damage to the brain stem.

In the management of encapsulated brain abscess, two events may occur necessitating immediate surgical interference: (1) development of spreading cerebral oedema with rapid loss of consciousness, or (2) rupture of the abscess into the ventricular system causing ventriculitis and meningitis, and requiring immediate removal of the focus of infection. The latter event is one of the chief causes of death due to brain abscess.

One of the most difficult problems in the management of brain abscess is the diagnosis of multiple abscesses. These account for the majority of surgical failures.

SEQUELAE

Epilepsy occurs in about 45% of survivors of cerebral abscess; in the majority of cases, however, this can be well controlled. Mental disorders and focal abnormalities such as aphasia, homonymous hemianopia, hemiparesis or sensory disturbances also occur.

CONCLUSION

A striking feature about intracranial suppuration in the present era of antibiotic therapy and improved surgery is that the incidence of such suppuration has not decreased. A decrease in incidence will only occur in the more careful management of the primary disorder and in the recognition that intracranial suppuration is a serious complication leading to possible death or neurological abnormalities. Prompt recognition and appropriate treatment will lead to a further reduction in mortality.

ACKNOWLEDGEMENT

I wish to express my gratitude to Dr. T. P. Morley for the time spent in reading the manuscript and for his many helpful suggestions.

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RADIOTHERAPY IN THE TREATMENT OF TESTICULAR TUMOURS

R. S. BUSH, 6T0

Introduction

MALIGNANT disease of the testis is relatively rare; in fact it comprises only 2% of tumours in males. However, these testicular tumours generally occur earlier in life than most:

Teratomas—average age of onset is 30 years.

Seminomas—average age of onset is 40 years.

The undescended testis is of particular interest since tumours are for more likely to occur: 1 in 80 develop a malignant tumour.[1]

Since most tumours of the testes are derived from a totipotential germ cell, their morphology and behaviour are more varied than almost any other tumour in human oncology. The resultant problems in therapeutic management are numerous.

Pathology

There are many differences of opinion with respect to the classification of tumours of the testicle. It is not within the scope of this article to discuss these, and the classification will be a simple division into two groups—seminomas and teratomas, the former being the more common.

Seminomas comprise 60% of malignant testicular tumours.[1] They are slow growing and markedly radiosensitive. Undescended testicles harbour this tumour more frequently than other forms of testicular tumour. The involved testis may be only slightly enlarged or may be ten times larger than normal, yet it usually maintains its normal contour. This is due to the tumour seldom invading the tunica covering the testicle.

The appearance of the neoplasm on gross section is greyish white, dense and homogenous so as to resemble a sarcoma. Not uncommonly yellowish and yellowish-brown areas of necrosis are evident. Cysts are never found.

Microscopically, the neoplasm is readily recognized by the monocellularity. The cells are moderately large, round or cuboidal, uniform in size and normally have distinct cell borders. The nucleus is large and centrally placed. Not uncommonly, the cells may be arranged in cords or have a tubular pattern. An admixture of lymphocytes is often present, and occasionally causes difficulty in interpretation because of the similarity to lymphosarcoma.

In a typical case of seminoma the primary tumour is present for six months to a year or longer before the symptoms cause the patient to seek medical advice.

Teratomas comprise about 40% of testicular tumours. This class of tumour may show no malignant characteristics for a considerable time. These tumours are variable in their colour, texture and may be cystic. They are fast growing, and metastasize much more quickly than seminomas. The tumour may attain a very great size.

The tumour probably arises from a germinal blastomere or primitive germ cell, and thus may contain structures derived from the embryonic layers. Thus, the microscopic appearance can be extremely varied. From the mesoderm there may be cartilage, bone, muscle, fat and lymphoid tissue; from the ectoderm there may be stratified epithelium; from the endoderm there may be attempts to form a primitive alimentary canal. The usual appearance is a mixture of lymphoid tissue, cartilage and tubular spaces giving the appearance of cysts.

Metastases

Successful treatment of cancer of the testis depends chiefly on control of metastases in the retroperitoneal lymph nodes. The lymphatic connection of the testicle due to its descent from the embryonic position near the kidney means that the first metastases are frequently in the lymph nodes along the abdominal aorta, and thence spread up this chain into the mediastinum and left supraclavicular node. However, with infiltration of surrounding tissues at the primary site, regional lymphatics may carry cells to the external and internal iliac and lower aortic nodes. The paraaortic nodes may have direct bilateral connections with the internal iliac nodes.[1]

As in other rapidly growing tumours, the blood stream is easily reached, and spread may occur this way and multiple secondaries reach the lung.

Seminomas metastasize later than the teratomas, and when they do, generally tend to go by way of lymphatics to the retroperitoneal lymph nodes. Teratomas metastasize much earlier, and normally much more extensively.

Diagnosis

Any swelling of the body of the testis should be held suspect. The differential diagnosis is among orchitis, torsion, epididymitis with spread to the corpus testis, gumma and haematoma of the testis.

Any hydrocoele in a young man should be tapped, especially if of recent origin and of rapid growth. If the fluid is blood stained, the suspicion of malignancy is heightened.

Radiographic examination of the chest should be carried out at the same time.

Assay of the patient's urine for gonadotrophic hormones may not give significant results in early cases of seminoma.

In all cases, the testicle should be surgically explored; if the doubt persists at operation the removal of the organ for histological study is justified.[1]

Choice of Treatment

Because of the tumour's marked radiosensitivity, irradiation plays a dominant role in the treatment of seminoma.

The plan of any treatment must be based on the distribution of testicular lymphatics. Metastases may extend from the pelvic nodes to the mediastinum with no clinical symptoms. Thus, as there is no surgical procedure radical enough to deal with such extensive communications, irradiation offers a much more effective alternative and can show considerably better results [1]

Simple orchidectomy offers the most certain method of dealing with the primary tumour.

Treatment of teratoma is not quite as straightforward as with seminoma. Teratomas are not so uniformly radiosensitive —some are more, but many are less so. Statistically, results of irradiation in teratomas are better than surgical lymphadectomy. In radiotherapeutic departments, teratomas would be managed similarly to seminomas. A few will require lymphadectomy, but this should be done only after the radiation treatment has been seen to have failed. The reason for not allowing surgery before radiation treatment is that the radiosensitivity of the malignant cells changes after disturbance from surgery.

Radiotherapy

It is accepted radiotherapeutic practice that as the volume of tissue which must be irradiated increases so the dosage must be decreased. Because the potential field of metastases, as outlined above, is a large volume, the dose has to be numerically small. However, due to the radiation sensitivity of testicular tumours, satisfactory treatment can still be given.

In patients with no clinical signs of metastases these areas must be irradiated:

(a) pelvis—external iliac nodes
internal iliac nodes
para-aortic nodes

(b) abdominal aortic and perirenal chains.

When evidence of metastases in the abdomen is present, the areas to be irradiated are:

(a) as above

(b) the whole of the chest and also the supraclavicular nodes.

In this case the time for total treatment would have to be increased due to the increase in tissue volume to be irradiated.

The dosage required to be reached in the treatment of testicular tumours may be compared to that for carcinoma of the cervix uteri. For the latter, the minimum dose is in the order of 7,000 r in 5 weeks over just part of the pelvis. In testicular tumour treatment, 2,000 to 2,500 r only are required in 5 weeks. Thus the much larger volume of tissue may be covered by the treatment.

Recurrence and Its Treatment

As the metastases from seminomas and some teratomas are often so radiosensitive, any that show up at a later date may be controlled and caused to recede. Recurrences are so likely due to non-irradiation of some areas to which the malignant cells may have spread that the patient must have regular and thorough examinations after the series of treatments is completed.

Note should be made here that even in radioresistant teratomas with wide metastasizing of the tumour, symptomatic relief may often be obtained with radiation.

Results

	Survived at least 5 years	
	Seminoma	Teratoma
Clinically free from metastases when first seen	80%	65%
Having metastases when first seen	30%	15%

Prognosis

The outlook for cases of testicular tumour, especially seminomas, treated before there is clinical evidence of metastases is most hopeful. When metastases are present, it is much worse, but there is still a good chance of a cure which makes radical radiation therapy worth while for any patient that is in reasonable condition.

Acknowledgement

The author is grateful to Dr. W. D. Rider for his helpful criticism in the preparation of this paper.

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CASE REPORT: SUBDURAL AND EXTRADURAL HEMATOMA

M. G. NORMAN, 5T8

MISS J. B., an 18-year-old student, was in a car accident at noon. Immediately following the accident she was unconscious for a short period of time. She was brought to the Emergency Department and on examination was found to be fully conscious, lucid, and there were no physical findings of significance.

Two and one-half hours after the accident there was slight deterioration in the level of consciousness. The patient was restless, but responded to her name and spoke lucidly. The pulse rate was 60 per minute and irregular.

In the following fifteen minutes the level of consciousness had deteriorated still further, only a moan being elicited in response to her name. The right plantar response had become equivocal.

During the next fifteen minutes the level of consciousness further deepened, and the patient responded only to painful stimuli. The left pupil began to dilate and its reaction to light became sluggish.

Over the next half-hour both pupils became dilated. The left pupil was fixed; the right was deviated externally and downwards. Corneal reflexes were present bilaterally, abdominals absent and planters bilaterally up-going. The pulse rate had dropped slightly to 56 per minute. The blood pressure was 115/88—unchanged from its original level. There were signs of a slight spastic right hemiplegia. Other significant findings at this time were blood behind the left eardrum and a puffy, egg shaped swelling in the left temporal region. Skull plates showed no fracture and the pineal was not calcified. At this time the patient was taken to the operating room as a neurosurgical emergency with a preoperative diagnosis of a left-sided extradural hemorrhage or acute subdural hemorrhage.

A burrhole in the left temporal region showed a moderate extradural haemorrhage. Following evacuation of this the brain remained "tight", and a burrhole in the right temporal region showed an acute subdural hemorrhage coming from a torn parasagittal vein. This vein was cauterized to stop the bleeding and the subdural hematoma evacuated.

Post-operatively the patient made a good recovery. Immediately following the operation the pupils were round, equal, and symmetrical, and reacted to light. The right-sided hemiplegia had disappeared.

The patient gradually recovered full consciousness, recognizing members of her family within 48 hours. The post-traumatic amnesia lasted for about 14 days, and the patient has continued to make a good recovery.

This case is presented because it illustrates:

1. The classic progression of signs,
2. The rapidity with which these signs may develop, and therefore,
3. The necessity of watching all patients closely following a period of unconsciousness,
4. The availability of the important clinical signs:
   —level of consciousness
   —pupils
   —blood pressure
   —pulse
   —neurological examination
5. The reason why bilateral burrholes are mandatory in exploring cases of suspected intracranial hemorr-

hage. (Although the combination of extradural and acute subdural is rare, bilateral subdural hemorrhages are not uncommon.)

The predominance of right-sided signs in this case is explained by the extradural hemorrhage on the left, or by pressure from the subdural hemorrhage on the right causing a Kernahan's notch.

ACKNOWLEDGEMENT

The writer wishes to thank Dr. W. Lougheed for permission to present this case and for help in preparation of this report.

CASE REPORT: RUPTURED UTERUS

J. A. WILKINSON, 5T8

MRS. R. C., a thirty-year-old Italian immigrant, first presented herself at the out-patient department of a Toronto hospital on Nov. 12, 1956.

Her obstetrical record revealed three full term normal deliveries in 1948, 1950 and 1952. None of the birth weights were known, and the 1952 delivery produced an infant who did not nurse well and died at 22 days, a diagnosis of cerebral damage being made.

The results of her pre-natal examination were as follows:

Past health—negative
Complaints and complications—none
Physical findings—no abnormalities
Weight gain—4 lb. (?)
Position—R.O.T. (last visit)
Hemoglobin—76%
Weight—125 lb.
Urinalysis—negative
Pelvic examination—not done

The expected date of confinement was established as Dec. 31, 1956.

The patient was admitted on Jan. 12, 1957 at one p.m. The admission physical examination revealed the following:

Head and Neck—clear
Chest—clear to percussion and auscultation
Heart—normal rate and rhythm
—B.P. 120/64
Abdomen—gravid
—position of foetus—R.O.A.
Urinalysis—slight trace of albumin.

At this time 5 mgm. of Vitamin K were given.

At two-thirty p.m. strong contractions were observed. The foetal heart was heard in the R.L.Q., and the position was established as R.O.T. The head, however, was noticed to be high. The cervix was 3½ to 4 finger breadths dilated with the membranes intact and bulging. Demerol 100 mgm. was administered.

At three-ten p.m. the membranes ruptured spontaneously. The foetal heart was still heard. A thin rim of cervix was present, and the head was still not engaged.

At four p.m. contractions were strong every two minutes, and the patient was pushing. The head was not engaged, and the foetal heart rate was 120 beats per minute. The cervix was fully dilated. The patient was X-rayed to assess pelvimetry. The report revealed a single foetus in the R.O.A. position. The pelvic inlet was small, and bony disproportion was suggested. An intravenous was started, and the patient was crossed and typed for blood. Atropine gr. 1/150 was given.

At five-forty-five p.m. the O.R. was phoned and a caesarean section was booked for six-thirty p.m. Demerol 100 mgm. was given. Deep ether was given to reduce the strength of contractions until the section could be done.

At six p.m., when ether was first given, the patient became restless, grasped at her abdomen and became semi-conscious. The foetal heart was no longer heard and uterine contractions ceased. Foetal parts were felt under the anterior

abdominal wall. The maternal pulse was 130 and the blood pressure 130/70. A catheter specimen of urine was stained with bright red blood A diagnosis of uterine rupture was made. The patient was taken to the O.R.

At six-thirty p.m. a lower abdominal mid-line incision was made, and the child was found totally outside the uterus. The placenta was partially outside, and approximately 800 cc. of blood were in the peritoneal cavity. The uterus was ruptured in the lower segment on the left side from the round ligament to within 2 cm. of the cervix. There was only moderate bleeding from the raw edges. Closure was decided upon and accomplished with two layers of chromic catgut with inversion of edges. Reperitonealization was done with a running suture. The abdomen was closed, and the patient was returned to her room in good condition.

The patient was supported during the operation and for the next two days with 1500 cc. of whole blood, I.V. glucose in distilled water and I.V. normal saline.

The autopsy report on the foetus revealed a still-born female weighing 6 lb. 9 oz. The lungs showed aspiration of amniotic fluid and intra-alveolar haemorrhage.

This case represents a spontaneous complete rupture of the uterus due to disproportion between a contracted pelvis and the foetal head. The fact that this followed three normal full term deliveries made its occurrence quite unexpected.

The etiology of rupture of the uterus can be classified as follows:

A. Spontaneous—sometimes with a predisposing factor of myometrial scarring caused by:
   i. previous section
   ii. myomectomy
   iii. curettage
   iv. perforation with a uterine sound
   1. Dystocia due to:
      a. a contracted pelvis
      b. abnormal presentation, e.g. transverse, brow, posterior, face
      c. pelvic tumor
   2. Excessive multiparity

B. Traumatic
   1. Instrumentation
   2. Manipulation
   3. Other trauma

C. Indiscriminate use of the oxytocic effect of posterior pituitary extract.

This patient also exhibits many of the classical signs and symptoms of ruptured uterus during labour. Those normally to watch for are:

1. Complaint of pain at the height of a contraction followed by cessation of labour.
2. Rapid, weak, thready pulse.
3. Laboured respirations.
4. Pale, cold, clammy skin.
5. Blood in a catheter specimen of urine.
6. Palpation of the uterus as a distinct mass beside the child free in the abdominal cavity.
7. Change in presentation or a recession of it at the cervix.
8. Signs of peritoneal irritation from free blood in the peritoneal cavity.

Beck claims that the prognosis in rupture of the uterus with complete tears approaches 100% maternal mortality unless the patient is quickly subjected to skilful surgery. The foetal mortality is also 100% in all but cesarean section ruptures.

Although in the treatment of this condition, hysterectomy is most commonly used, in this case, it was felt that the tear could be sutured and the uterus saved.

It is interesting to note that the patient returned to hospital almost one year later to the day, and gave birth to a normal child by an uncomplicated cesarean section.

Reference:

Beck: Obstetrical Practice, 6th Edn.

PRIMARY GLAUCOMA

V. H. CHROM, 5T8

Definition

GLAUCOMA is a disease characterized by unstable or increased intraocular pressure, usually causing impairment of vision ranging from slight abnormalities such as blurring of vision, "halos" and visual field defects to absolute blindness.

Classification

Glaucoma may be divided into two types: *primary* glaucoma in which the etiology is not well understood, and *secondary* glaucoma in which the etiology consists of various well known eye conditions, for example, uveitis, corneal perforation, intraocular haemorrhage, intraocular tumors and dislocation of the lens.

This paper will deal only with the primary type of which there are three subdivisions.

I. Infantile Glaucoma

etiology and pathogenesis

This condition is also known as Buphthalmos or Hydrophthalmos. It is a congenital anomaly resulting from partial or complete failure of development of the drainage channels at the angle of the anterior chamber. The canal of Schlemm may be deficient or absent, and there are no aqueous veins. The aqueous humor must return to the venous system via the ciliary venous plexus and the veins of the iris. This is not an efficient mechanism; hence, the intraocular tension rises.

Since the coats of the eye in the infant are not rigid, the cornea and sclera distend, and the eye becomes quite large. The cornea becomes globular, and deep cupping of the optic disc occurs.

incidence

Infantile glaucoma occurs once in every two thousand births. It is twice as common in males as in females. About 90% of cases become evident in the first six months of life.

The disease has an hereditary tendency, and is often associated with other congenital anomalies, especially facial nevi and multiple neurofibromatosis (Von Recklinghausen's Disease). Both eyes are usually affected (75% of cases).

signs and symptoms

The obvious sign is an abnormally large eye. The sclera is bluish. The cornea is globular, and the anterior chamber is therefore very deep. Episodes of increased pressure are manifested by irritability, rubbing of the affected eye and photophobia. In these cases there is circumcorneal congestion and a steamy cornea. The lens is flattened and displaced backwards resulting in tremulousness of the iris (iridodonesis). The eye is usually myopic and astigmatic due to its expansion and moulding.

Although raised tension is often minimal, if present at all, deep cupping and optic atrophy usually take place in the untreated eye with resultant loss of vision.

diagnosis

A large eye and a globular cornea are also seen in the congenital anomaly: keratoglobus. In this condition, however, the cornea is clear and the angle, optic disc and intraocular pressure are all normal.

treatment

Miotics such as Pilocarpine and Physostigmine are of little value, but should be

tried; they may be effective if the obstruction to drainage is incomplete.

Surgery is usually necessary. The measures employed are either filtration operations or attempts at reopening the drainage channels and the angle of the anterior chamber.

PROGNOSIS

The treatment of infantile glaucoma is notoriously unsatisfactory, probably because too much damage has unavoidably occurred prior to diagnosis and treatment. However, if surgery is instituted before there is too much stretching of the corneo-sclera coat, the prognosis for control of tension and maintenance of vision is quite fair.

II. Closed-angle Glaucoma

ETIOLOGY AND PATHOGENESIS

The most important factor in the development of closed-angle glaucoma appears to be the existence of a narrow anterior chamber angle. A narrow angle usually co-exists with a shallow anterior chamber. Factors which make for shallowness of the anterior chamber and a concomitant narrow angle include heredity, advancing age and hypermetropia.

In an eye with this anatomical configuration, dilatation of the pupil or congestion of the ciliary body may result in the root of the iris coming against the cornea and blocking off the angle. Drainage of aqueous is thus interfered with, and the intraocular tension rises.

The acute congestive attacks in this type of glaucoma are probably not directly due to a rise in intraocular tension, but rather to a liberation of histamine-like substances from damaged tissues. Damage to tissues resulting in this process is brought about by circulatory strangulation consequent upon increased intraocular pressure.

The anatomical explanation is, however, not quite adequate to explain why closed-angle glaucoma occurs most commonly in excitable people and why it does not occur in every person with a narrow angle. The answer is that in order to develop closed-angle glaucoma an additional factor must probably be present, namely, vasomotor instability. It is not a case of the patient being normal with only his anterior chamber angle being abnormal. Both the patient and his eye are abnormal; as Duke-Elder puts it, in closed-angle glaucoma there is "a sick eye in a sick body". When this type of glaucoma is treated surgically, the structural defect is corrected, but the functional vasomotor instability persists, and the operation merely renders the latter innocuous inasmuch as it can no longer produce rises in tension.

INCIDENCE

This disease which is still referred to in most textbooks as "Acute Glaucoma" or "Acute Congestive Glaucoma" occurs in about 0.2% of the population. It is seen chiefly in the 40 to 60 age-group, more often in females, and especially in emotional, excitable people who often have other evidence of vasomotor instability. It is almost always associated with a narrow anterior chamber angle. Eyes in which this type of glaucoma develops are often small and hypermetropic with a relatively large lens. Since both eyes are usually anatomically similar, closed angle-glaucoma is usually bilateral although one eye is usually affected before the other.

SIGNS AND SYMPTOMS

The disease may be divided into five stages:

1. *Prodromal Stage*

This is characterized by intermittent episodes of blurring of vision, the appearance of halos around lights, and occasional headaches. The blurring and halos are due to corneal oedema which is in turn due to impairment of the circula-

tion of the aqueous. During the attacks the intraocular tension may rise to 40 to 60 mm. of mercury, but the eye remains white.

The attacks are usually associated with emotional factors or with other factors causing pupillary dilatation such as darkness. They are particularly common following visits to the cinema or watching television. In between attacks the eye is perfectly normal.

2. *Stage of Constant Instability*

In this stage the attacks of blurring and halos occur every night. The diurnal variation of the intraocular tension becomes markedly increased over the normal value of 2 to 5 mm. of mercury, and may be as high as 60 mm. of mercury. The tension tends to fall during sleep.

3. *Acute Congestive Attack*

A fulminating attack occurs sooner or later after the stage of constant instability is reached. It is the chief non-traumatic ophthalmological emergency. It may be precipitated by an emotional upset.

The acute attack is characterized by a sudden onset of severe pain located mainly in the eye, but felt over the entire distribution of the trigeminal nerve. There may be vomiting and even collapse. In addition there is blurring of vision and a rapid diminution in visual acuity taking place in the course of a few hours. In some cases both eyes are affected simultaneously.

On examination a red eye is seen due mainly to circumcorneal congestion. The cornea is cloudy, and the anterior chamber is very shallow. The pupil is semi-dilated, oval, and does not react to light or accommodation. The intraocular tension is raised, and the eye feels stony hard.

The attack may resolve spontaneously, but unless treatment is instituted early, it may progress to total abolition of vision.

4. *Stage of Chronic Congestive Glaucoma*

This stage develops following repeated acute congestive attacks. The visual acuity is considerably reduced. There is concentric constriction of the visual fields, and the typical scotomata seen in simple glaucoma appear as well as cupping of the optic disc. The intraocular pressure becomes permanently elevated chiefly due to permanent narrowing of the angle by formation of peripheral synechiae. There is very little difference between chronic congestive glaucoma and advanced simple glaucoma as far as the clinical picture is concerned.

5. *Stage of Absolute Glaucoma*

If the disease is not treated, the patient eventually becomes completely blind. The cornea is clear but insensitive, and may develop vesicles or filaments. The pupil becomes dilated, grey or greenish, and is surrounded by a broad zone of pigment. There is pronounced cupping of the optic disc. The intraocular pressure is at first very high, and the eye is painful. Eventually the sclera gives way due to the pressure; it becomes scarred, and the eye increases in size. This is one cause of the intraocular pressure now diminishing; the other cause is degeneration of the ciliary body which takes place at this time. The eye may then shrink; commonly corneal ulceration takes place followed by hypopyon, panophthalmitis and phthisis bulbi.

DIAGNOSIS

It is extremely important to diagnose closed-angle glaucoma in the prodromal stage because once an acute congestive attack has occurred, irreparable damage has already been done.

A history of halos may also point to early cataractous lenticular changes; the two conditions may be differentiated by Finchman's test using a stenopoeic slit.

Observation of a rise in tension is difficult because between attacks the ten-

sion is normal. However, provocative tests may be employed which in cases of latent glaucoma will demonstrate abnormal rises in intraocular tension. Commonly used are the dark-room test, the water-drinking test and the mydriatic test. All these tests must be used with great care as they may precipitate acute congestive attacks with which the ophthalmologist must be prepared to deal immediately. The tests are positive in 50% of cases, but when negative, the presence of prodromal acute glaucoma can still not be excluded.

Shallowness of the anterior chamber may be observed by direct inspection or by the use of the slit lamp. The configuration of the angle may be ascertained by gonioscopy.

Because of the haziness of the cornea in acute congestive glaucoma, it is impossible to do a good ophthalmoscopic examination. The haze may be overcome by the instillation of a drop or two of glycerine into the eye.

In the acute congestive attack and chronic congestive stage the diagnosis is not difficult to make. The differential diagnosis of acute congestive glaucoma is that of the Red Eye (acute conjunctivitis and acute iritis). It is extremely important to differentiate acute glaucoma from acute iritis because the treatment of the two is diametrically opposed. Unfortunately, mistakes have been made in practice, and mydriatics which are the treatment for iritis have been instilled into acute glaucomatous eyes resulting in very considerable aggravation of the condition. For this reason some points in the differential diagnosis of the Red Eye are listed in the following table:

	CONJUNCTIVITIS	IRITIS	GLAUCOMA
Injection	superficial	deep ciliary	deep ciliary
Secretion	muco-purulent	watery	watery
Pupil	normal, brisk light reaction	small, irregular, fixed	large, oval, fixed
Media	clear	pupillary opacities sometimes	corneal oedema
Tension	normal	usually normal	raised
Pain	absent to mild (eye only)	moderate (eye, V^1)	severe (eye, V^1, V^2, V^3)
Tenderness	absent	marked	marked
Vision	good	fair	poor
Onset	gradual	usually gradual	sudden
Systemic complications	absent	little	may be extreme with prostration and vomiting

TREATMENT

1. *Prodromal Stage*

Miotics such as Pilocarpine 0.5 to 5.0% or Physostigmine 0.25 to 1.0% should be instilled into the eye as often as is necessary to control the attacks. Prophylactic instillation is indicated whenever excitement or darkness are anticipated.

If the miotics are successful in completely controlling the halos, and if there is no evidence of increasing impairment of vision, the patient may continue to use them under supervision indefinitely.

If the disease is not checked in spite of the regular use of miotics, or if the patient fails to apply the drops regularly, or if he is unable to have regular medi-

cal supervision, prophylactic iridectomy should be undertaken. When this is done in the prodromal stage, the prognosis is very good.

2. *Acute Congestive Attack*

Although surgery must frequently be resorted to, an attempt should first be made to control the condition by medical measures.

Bed rest and full sedation with morphine if necessary should be employed. Physostigmine combined with Pilocarpine should be given every ten minutes for the first hour and then every 20–30 minutes until miosis is attained. Local heat (hot bathings or diathermy) increases the effectiveness of the miotics. Diamox 500 mgm. per os initially followed by 250 mgm. q. 4h. works presumably by inhibiting secretion of the ciliary body. Retrobulbar injection of adrenalin and novocaine is sometimes used.

If these measures fail to reduce the tension in 12 to 24 hours, a basal iridectomy should be performed.

If medical management is successful in relieving the attack, there is some difference of opinion as to whether or not elective surgery should be undertaken on the quiet eye. Some feel that continued medical management with miotics is acceptable; however, the majority believe that surgery should always be performed on an eye that has sustained an attack of acute congestive glaucoma. The recommended operation is iridectomy if peripheral synechiae are not extensive; otherwise, a filtering operation (iridencleisis) is done. Duke-Elder goes so far as to say that the other eye should also be operated upon (prophylactic iridectomy) as acute glaucoma very commonly occurs in it sooner or later. In any case, miotics should be instilled into it twice daily.

3. *Absolute Glaucoma*

The treatment of choice is excision of the eye. If the patient refuses this, cyclodiathermy may be undertaken. Otherwise, medical management may relieve the pain.

PROGNOSIS

If treatment is started in the prodromal phase, there is as yet no impairment of visual acuity, and provided medical measures are adhered to carefully, or surgery is undertaken, the prognosis is good.

The prognosis of an acute congestive attack depends largely on the speed and vigour with which treatment is instituted. Although some impairment of vision is almost inevitable, it is still possible to achieve fairly good function in many cases following iridectomy.

Even in the chronic congestive stage, much can still be achieved, with miotics and surgery, in salvaging vision.

Once the stage of absolute glaucoma has been reached, the complications of pain and ocular inflammation are very likely to occur. Malignant change is also not uncommon, so that removal of the eye should be strongly advocated.

III. Simple Glaucoma

ETIOLOGY AND PATHOGENESIS

The angle of the anterior chamber may be of any type in simple glaucoma: wide, medium or narrow; thus its configuration does not enter into the etiology at all, and it is wrong to refer to the condition as "wide-angle glaucoma", as some textbooks do.

The important factor in simple glaucoma is probably an impediment to drainage of the aqueous in the region of the canal of Schlemm, possibly in the trabeculae, which raises the intraocular pressure. The raised pressure probably plays an important role in the production of cupping of the disc and development of visual field defects due to pressure atrophy of parts of the retina.

But here again, the impeded drainage factor in itself is insufficient to account

for all the facts. There is often very little correlation between the rise in intraocular tension and the development of cupping of the optic disc and visual field defects. One can have rapidly developing scotomata with a complete abscence of raised tension. In other cases, the visual fields may continue to deteriorate even after tension is relieved surgically. In addition, we know that persistently increased intraocular pressure is quite compatible with normal discs and fields because this occurs in some cases of secondary glaucoma.

There is probably another etiological factor operating in simple glaucoma, that of vascular sclerosis. It is true that simple glaucoma occurs in the age-group of arteriosclerosis, and that there is usually other evidence of arteriosclerosis in patients with simple glaucoma. It is believed that due to vascular sclerosis there develops an inadequacy of the blood supply to certain vital portions of the eye, particularly the optic disc and the posterior portion of the retina. The development of the impediment to drainage at the angle of the anterior chamber may somehow or other be caused by the same arteriosclerotic process. Whatever the process is at the angle, it alone is probably responsible for the raised intraocular pressure.

Although the intraocular pressure is raised in the vast majority of cases of simple glaucoma, there are some cases with typical cupping and field defects in eyes in which the intraocular pressure is perfectly normal. Such cases may perhaps be explained by vascular sclerosis occurring at the posterior pole of the eye, but being absent or insignificant at the angle.

INCIDENCE

This type of glaucoma is about ten times more common than closed-angle glaucoma, occurring in about 2% of the population. There is a 10% hereditary incidence. It is seen chiefly in the 60 to 70 age group, but is quite common in younger middle-aged people. There is no particular sex incidence and no particular associated psychological factor. Simple glaucoma is almost always eventually bilateral.

The disease progresses very insidiously producing only mild headaches and eyeaches in some cases. The condition may thus be far advanced before the patient seeks treatment. However, an observant, intelligent patient may seek medical advice if he notices a visual field defect or a decrease in his ability to adapt to darkness, or if he finds that he needs his glasses changed frequently and that such changes are never quite satisfactory.

In early simple glaucoma there are three characteristic signs: unstable or raised intraocular tension, cupping of the optic disc and visual field defects.

The disease, if unchecked, may or may not develop a chronic congestive stage with impairment of central vision. In all cases, the end result is complete blindness—absolute glaucoma—just as occurs in untreated closed-angle glaucoma.

DIAGNOSIS

The diagnosis in the early stages depends on demonstration of the three characteristic signs.

1. *Tension*

As in closed-angle glaucoma, the initial disturbance is an exaggeration of the normal diurnal variation of intraocular pressure rather than a protracted rise in tension. To measure this, tonometry is required, and it is preferable to admit the patient to hospital for 24 hours so that repeated readings may be taken. Later in the course of the disease, this will usually not be necessary because of the presence of permanently raised intraocular pressure. This, however, is not always the case; severe damage to the eye may take place in the absence of per-

manently raised tension. Suspicion of glaucoma should, therefore, be aroused whenever the diurnal variation in intraocular tension is greater than 5 mm. of mercury even though the normal upper limit of the intraocular pressure (25 mm. of mercury) is not exceeded.

It is of interest to note that in simple glaucoma, the maximum elevations of tension usually occur in the morning and afternoon with a fall in the evening, whereas in closed-angle glaucoma the maximum rise is in the evening.

Provocative tests used here are the water-drinking test, the venous congestion test, and the bulbar pressure test.

2. *Cupping of the Optic Disc*

Care must be taken to differentiate glaucomatous cupping from a deep physiological cup. This is often difficult. Important differential points are:

a. The glaucomatous excavation reaches the edges of the optic disc; the physiological cup does not.
b. The edges of the glaucomatous cup are steep, not gradually sloping.
c. There is always some optic atrophy present in a glaucomatous excavation so that the disc is pale and the markings of the lamina cribrosa are easily seen.
d. The retinal vessels end abruptly at the disc margin, and their continuations in the floor of the cup are out of focus in glaucoma.

Another condition which is very difficult to differentiate from a shallow glaucomatous cup is the slight depression which occurs in primary optic atrophy. The important point here is the colour of the disc: the shallow glaucomatous cup is pink, whereas the cup of primary optic atrophy is white.

The etiology of cupping is probably dual: raised pressure, acting mechanically, and arteriosclerosis, causing ischemic atrophy of the nerve head.

3. *Visual Field Defects*

It is important to chart the fields repeatedly in assessing progress of the disease because central vision remains unimpaired until the condition is well advanced, and therefore, the subjective impression of the patient that his vision is not deteriorating cannot be relied upon. Charting the visual fields may be done quite adequately by confrontation testing or the use of a tangent screen, but for accuracy, perimetry is required.

The first change is an enlargement of the blind spot to form a sickle-shaped scotoma with its concavity directed towards the point of fixation and usually facing downwards (Seidel's sign). The scotoma then spreads to gradually involve the whole upper nasal field. At this stage the scotoma has a characteristic sharply defined horizontal edge (Roenne's step). Then arcuate scotomata spread peripherally and temporally, like arms, from the nasal scotoma. These meet, and there remains central vision plus a temporal remnant of the field. Finally central vision is lost, and although the paracentral patch of the temporal field may persist, for practical purposes the eye is blind.

The peculiar pattern of these changes is probably due to the fact that many of the optic nerve fibres are crowded on the temporal side of the optic disc, and run an arcuate course to the macula. It is in the region of these fibres—the posterior pole of the eye—that the effects of glaucoma first become apparent.

TREATMENT

1. *In the Early Stages*

As in closed-angle glaucoma, miotics are the first line of attack. Strength and frequency of administration will vary with the individual patient, and should be regulated by tonometric control.

Diamox 250 mgm. q.4–6h. is helpful in some cases of more advanced glau-

coma, especially in the patient who is a poor operative risk.

Medical measures are successful in controlling many cases, and may be employed indefinitely as long as the base intraocular tension is kept below 30 mm. of mercury, and there is no further deterioration in vision.

The indications for surgery are the same as in the prodromal phase of closed-angle glaucoma: failure to control the disease by medical means, failure of the patient to use his drops regularly, or inability or neglect of the patient to follow regular medical supervision.

The operations of choice are those which establish a "filtering scar" in the sclera through which the aqueous may drain. These include Elliot's trephining, Lagrange's operation, Herbert's operation, and iridencleisis. Other surgical measures include cyclodialysis and cyclodiathermy.

Sometimes, with advanced visual field loss, surgery may cause sudden complete loss of vision, but this is rare, and on the whole more eyes are lost by delay in operating than by surgical intervention.

2. *In the Chronic Congestive and Absolute Stages*

The treatment is the same as in the corresponding stages of closed-angle glaucoma.

PROGNOSIS

The initiation of treatment in the early stages can often check the progress of the disease even though it cannot restore that vision which is already lost. If medical treatment is effective in controlling the condition, the chances that it will continue to do so indefinitely are fairly good.

If, however, surgery must be resorted to, the prognosis is uncertain. Surgery can only help to relieve intraocular tension; it obviously does not affect vascular sclerosis so that vision may continue to deteriorate even after surgery. In such cases, miotics should be continued for life.

It is true that in general the earlier medical or surgical measures are instituted in simple glaucoma, the better the prognosis for preservation of visual function.

The prognosis in the chronic congestive and absolute stages is the same as in the corresponding stages of closed-angle glaucoma.

PROPHYLAXIS OF ADULT PRIMARY GLAUCOMA

Glaucoma is a common disease, affecting 2% of the population, and a leading cause of blindness. One out of every ten glaucoma sufferers becomes blind. One out of every eight blind persons in Canada and the U.S.A. is a victim of glaucoma. Much of this blindless is needless and preventable.

It has been repeatedly stressed in this paper that in both types of primary glaucoma, the earlier treatment is sought and initiated, the better the prognosis. It is, therefore, important that the general public be made increasingly aware of the glaucoma problem, and that they be encouraged to seek treatment early if they have any symptoms suggestive of chronic glaucoma of either type. These symptoms are:

1. Frequent changes of glasses, none of which seems quite satisfactory.
2. Deterioration in the ability to adjust the eyes to darkness as when watching motion pictures or television.
3. Loss of side vision.
4. Blurring of vision.
5. Seeing halos around lights.

However, in view of the fact that it is quite possible to have early simple glaucoma without any of these symptoms, the best defense against glaucoma is a thorough eye examination at least once

every two years after the age of 40. If the general public employ this measure, there will be a significant reduction in blindness due to glaucoma.

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AURICULAR ARRHYTHMIAS

J. H. FOWLER, 5T8

"I dined with Dr. M. Hall, and was indebted to him for one of the grandest spectacles that human eye can behold. I witnessed the circulation in the web of a frog under a microscope of one hundred magnifying power. I know not whether you have ever seen this or not; if not, you have no conception of its beauty. With what ideas does it fill the mind!"

James Jackson, Jr.
(in a letter to his father, Oct. 1, 1832)

IRREGULARITIES of cardiac rhythm have commanded the interest and imagination of lay people and scientists alike throughout the centuries. Only toward the end of the nineteenth century, however, were apparatus and techniques devised to permit accurate analysis and classification of the different types of arrhythmias. Indeed, even so comparatively innocent an irregularity as the premature beat (extrasystole) was seriously enough viewed by the profession that countless patients were subjected to prolonged, unnecessary treatment, and excluded from admission into the services, military and civil, as late as 1913 because of the vague prognostications of danger accompanying detection of this arrhythmia.

The classical work on arrhythmias was performed by Sir Thomas Lewis some thirty-odd years ago, and until the past decade his work went unchallenged. From 1947–1951 Prinzmetal's group at U.C.L.A. employed high speed cinematographic techniques and the cathode-ray oscillograph (neither of which was available in Lewis' time) to establish firmly the *ectopic focus* theory as opposed to Lewis' *circus* theory to explain the physiology of the four auricular arrhythmias, and to demonstrate that all four had a common etiology. He created each arrhythmia experimentally by placing a drop of aconitine on the atrial wall, and meticulously documented the muscular action with full colour, slow motion magnified photography.

Classification (Best and Taylor)

A. Due to impaired conduction through the A-V bundle and its ramifications
   1. Stem of bundle
      a. Delayed conduction
      b. Partial heart block (missed beats)
      c. Complete heart block
   2. Bundle branch block (intraventricular block)

B. Wolff-Parkinson-White syndrome

C. Due to abnormal impulse formation
   1. Premature beats (extrasystoles)
      a. Ventricular
      b. Nodal
      c. Auricular

2. Paroxysmal tachycardia
   a. Auricular
   b. Nodal
   c. Ventricular
3. Auricular flutter
4. Auricular fibrillation
5. Ventricular fibrillation

D. Alternation of the heart

E. Due to vagal influences
1. Sinus arrhythmia
2. Phasic irregularity
3. Sinus bradycardia
4. Sino-auricular block
5. Auriculo-ventricular block

General

The sino-auricular node, at the junction of the superior vena cava and right auricle, is the "pace-maker" or originator of the cardiac activity impulse. From here, the impulse spreads out through the auricular muscle fibres which conduct it to the atrio-ventricular node in the wall of the right auricle at the lower part of the inter-auricular septum. The auriculo-ventricular bundle of His carries the impulse down into the interventricular septum, and then divides into right and left bundles which continue on as the arborizing subendocardial network of Purkinje from which finer fibres extend outwards to supply the muscle syncytium itself.

In the ectopic arrhythmias discussed below, the "pace-maker" is temporarily of more protractedly subordinated to a hyperactive discharging focus in the myocardium of the ventricles, auricles, or even in the A-V node itself.

1. Premature Beats (Extrasystoles)

incidence

The commonest of all arrhythmias, the premature beat is likely to have occurred in everyone at one time or another. It is common in older folk and exceptionally rare in infants and children. Men are more frequently troubled by the symptomatic forms, but the sex difference is not marked (123 males to 95 females in one series).

etiology

The cause is unknown, but symptoms in many cases occur in conjunction with anxiety, gastric upset, over-indulgence and fatigue. Occasionally specific precipitating factors such as smoking are apparent from the history, and rarely special forms e.g. pulsus bigemini, may reveal digitalis overdose.

The focus most frequently arises in the ventricular myocardium, but when it arises in the auricle, the tendency toward later development of flutter and fibrillation is said to be much greater. Differentiation between ventricular and auricular types usually requires an electrocardiogram.

symptoms

Statements such as "my heart skipped a beat" or "it seemed to turn over" convey the sensation of a beat out of turn with a following pause which characterizes most symptomatic cases. At least 50%, however, have no symptoms whatever.

Occasionally sharp pain over the apex may be felt at the time of the premature beat; but whereas most of these have no heart disease, in a few, with rapidly repeated premature beats, the pain may be related to early coronary artery disease.

Faintness may be noted with rapid, frequent premature beats.

signs

The common finding on auscultation is the typical early diastolic premature beat, followed by a pause, and then resumption of normal rhythm, the first beat being louder.

Multiple premature beats may occur in rapid succession without normal beats between them. Rarely a true extrasystole

may occur i.e. a ventricular contraction interpolated between two normal beats.

DIFFERENTIAL DIAGNOSIS

Atrial fibrillation may be confused with rapid premature beats, but exercise tends to abolish the irregularity in the latter, whereas it makes the irregularity more pronounced in the case of fibrillation.

In rare cases E.C.G. tracings may be required in distinguishing premature beats from partial heart block and gross sinus arrhythmia.

TREATMENT

As the prognosis is almost uniformly excellent, an asymptomatic patient should not even be informed of his arrhythmia. In most symptomatic cases, reassurance and hygienic measures (regular rest, moderate diet, moderate living) permit reduction or complete relief of symptoms.

Occasionally an apprehensive patient may obtain much relief from daytime sedation (phenobarbitone gr. $\overline{ss}$ q. i. d.).

In the rare circumstance in which the patient is rendered a semi-invalid by repeated attacks accompanied by faintness and precordial pain, quinidine sulphate 0.2 Gm. t. i. d. p. o. or (for ventricular premature beats) procaine amide 0.5 Gm. q. 6h. p. o. with or without phenobarbitone often provide relief.

II. Paroxysmal Tachycardia

INCIDENCE

Although not as common as premature beats, paroxysmal tachycardia is certainly the next most common disorder of rhythm, for a large proportion of those afflicted are mild cases or even asymptomatic. The auricular variety, occurring most frequently in younger patients without heart disease, is six times as common as its ventricular counterpart which is usually associated with coronary artery disease.

ETIOLOGY

The ectopic focus usually arises with no apparent provocation although occasionally fatigue, indigestion, tobacco, alcohol, digitalis poisoning, infection or heart disease may be implicated. Cardiac infarction is the most frequent accompaniment of ventricular tachycardia, while thyrotoxicosis and mitral stenosis are rarely associated with an auricular tachycardia which may go on to fibrillation.

SYMPTOMS

1. Palpitation or flutter in chest of *sudden* onset and cessation.
2. Dyspnea.
3. Angina pectoris.
4. Syncope and even convulsions may occur with the more rapid rates.

SIGNS

1. *Regular* heart rhythm, 150–250 per minute usually, of sudden onset.
2. No variation of rate with emotion or exercise.
3. With ventricular tachycardia, as opposed to auricular, the rhythm may be slightly less regular, the first heart sound will vary in intensity, and other manifestations of heart disease may be noted. An E. C. G. is occasionally required to differentiate these two.

COMPLICATIONS

1. Congestive heart failure with prolonged attacks (as long as ten days).
2. Myocardial infarction.
3. Angina Pectoris.
4. Syncope.
5. Conversion of ventricular tachycardia to ventricular fibrillation and death.

DIFFERENTIAL DIAGNOSIS

Extreme sino-auricular tachycardia has a gradual onset, varies with exercise and emotion, and usually has a readily demonstrable cause e.g. shock.

Auricular flutter is manifested usually as a slower tachycardia (75, 100, 125), and vagal stimulation is followed by an irregular return to the previous heart rate. Flutter is usually a long-term disorder rather than a paroxysm, and is seen to include A-V block on E. C. G. inspection.

Auricular fibrillation is irregular.

TREATMENT

Short attacks (of a few minutes duration) without debilitating symptoms should be treated by elimination of the provoking stimulus if it can be found, and assurance to the patient that his disorder is a common one occurring in healthy people without heart disease.

To stop an attack, attempts to stimulate the vagus nerve mechanically should be made. These include massaging the right carotid body for twenty seconds, applying pressure to one eyeball for a similar period, and the manoeuvres of Valsalva and Muller (expiring and inspiring forcibly against a closed glottis respectively). These procedures either have no effect or cause a sudden return to normal rhythm which is maintained after the manoeuvre is stopped.

If these measures fail, drug therapy may be employed:

1. 0.4 Gm. quinidine sulphate orally with a sedative e.g. 0.2 Gm. nembutal. On awakening from sleep 95% of patients will have reverted to normal rhythm.

2. A more prolonged quinidine course may be required: 0.4 Gm. q. 2h. x 5 daily p. o.

3. In infants where extreme tachycardias are likely to bring on cardiac failure, normal rhythm is nearly always secured by digitalization with 0.5 mgm. Cedilanid intramuscularly, later doses being governed by clinical response. Digitalization is occasionally effective in terminating adult attacks as well.

4. For ventricular tachycardia, procaine amide 0.5 Gm. q. 6h. is usually effective.

Prophylaxis is a difficult task. General measures of a moderate living pattern with adequate sleep combined with drug therapy in troublesome cases seem to offer the best hope of success. A suggested regimen is quinidine sulphate 0.2 Gm. t. i. d. or procaine amide 0.25 Gm. q. i. d.

III. Auricular Flutter

INCIDENCE

The least common of the arrhythmias, flutter occurs about twenty times less frequently than auricular fibrillation. Males are affected three times as frequently as females, and 75% of cases are seen in the over-forty age group although it is seen occasionally in children and even infants.

ETIOLOGY

Prinzmetal contends that, as with the previously discussed arrhythmias, single or multiple ectopic foci, discharging in this case more rapidly than in the other two, are responsible for the greatly increased auricular rate (250–350 per minute).

Heart disease—chiefly rheumatic heart disease with mitral stenosis, hypertension, coronary artery disease and thyrotoxicosis—is seen as a frequent accompaniment of this disorder; less frequently, flutter may occur in a healthy heart.

Precipitating factors include sudden effort, excitement, trauma and surgical procedures in the chest.

SYMPTOMS

Palpitation of sudden onset is noted similar to that described in paroxysmal

tachycardia, but here the paroxysms are much longer, often lasting days, months and even years. Less than 25% of cases of flutter are permanent i.e. non-paroxysmal.

Symptoms of congestive heart failure may be precipitated by the onset of flutter, especially in those frequent cases associated with serious heart disease.

SIGNS

1. Regular tachycardia, usually about 160 per minute.
2. Jugular venous pulsations considerably faster than 160 per minute.
3. E. C. G. shows 2:1 A-V block in 50% of cases, others ranging up to 6:1.
4. Carotid body massage frequently produces sudden halving of the rate which returns to the original flutter rate. upon release of pressure.

DIFFERENTIAL DIAGNOSIS

Sino-auricular tachycardia begins more gradually, and varies with exercise *gradually*. Exercise often doubles the ventricular rate *suddenly* in auricular flutter.

Paroxysmal tachycardia is of shorter duration, usually in the absence of heart disease, and the E. C. G. demonstrates no A-V block.

Atrial fibrillation is more irregular, and is characterized by specific E. C. G. changes (auricular rate 350–600).

TREATMENT

Rare attacks of short duration in the absence of underlying disease may need only reassurance.

Digitalization results in fibrillation which, upon stopping the digitalis, reverts to normal rhythm in from 30-50% of cases. In most cases A-V block is increased resulting in a slower ventricular rate.

In digitalized patients with fibrillating auricles, quinidine sulphate 0.2 Gm. t.i.d. usually slows the auricles, and occasionally produces reversion to a normal rate.

IV. Atrial Fibrillation

INCIDENCE

Ranking third behind premature beats and paroxysmal tachycardia, fibrillation ranks first in importance, for the frequently associated complications may be effectively prevented by adequate therapy. The incidence increases with each decade in the patient's age, and males outnumber females in the proportion of 7:3. The rare cases of paroxysmal fibrillation occurring in young healthy people may occasionally be seen in families.

ETIOLOGY

An ectopic focus discharging at rates of upwards of 350 per minute initiates the impulse in fibrillation, but the exact cause is unknown.

Mitral stenosis is the most frequent accompanying condition, followed by hypertension, coronary artery disease, thyrotoxicosis and senile heart disease. It is exceptionally rare in the presence of angina pectoris or subacute bacterial endocarditis.

Paroxysms may be associated with excesses of alcohol or tobacco, and are occasionally initiated in the course of an acute infection such as pneumonia.

SYMPTOMS

No symptoms may be noted in long-standing cases or those with ventricular rates in the normal range.

Palpitation is the commonest complaint, often with a discomforting, fluttering feeling in the neck.

Dyspnea and other symptoms of cardiac failure may supervene if underlying heart disease is severe.

SIGNS

1. An irregularly irregular heart rhythm is the characteristic auscultatory finding, with the rate ranging from 75–130 usually.

2. A pulse deficit between the radial and auscultatory figures is often noted.

3. Random variation in intensity of heart sounds.

4. E. C. G. and phlebograms confirm the varying A-V block with fibrillating auricles.

COMPLICATIONS

Sudden death of unexplained origin may occur at any time. Embolic phenomena are seen in about 20% of cases. Cardiac failure may be precipitated by the onset of a paroxysm.

DIFFERENTIAL DIAGNOSIS

Gross sinus arrhythmia rarely has associated heart disease, and an E. C. G. dispels all doubts.

Multiple premature beats become less marked with exercise, whereas in fibrillation, the heart rhythm becomes still more irregular.

Paroxysmal tachycardia shows no heart block on E. C. G., and has a regular ventricular response on auscultation.

Atrial flutter with variable block requires an electrocardiogram to differentiate it from atrial fibrillation.

TREATMENT

In patients with severe heart disease, the aim should be to decrease the ventricular rate and improve cardiac function. The drug of choice is digitalis, or in more severe cases digoxin intravenously (1 mgm. stat, 0.5 mgm. after 6 hours, and 0.25 mgm. orally thereafter).

In patients in good health, an attempt to revert the fibrillation to normal rhythm may be considered after digitalization by employing quinidine sulphate in doses of 0.4 Gm. q. 2h. x 5 daily, and within three days 60-70% of cases may be expected to revert to normal. Digitalization prevents an excessive ventricular rate increase while the auricular rate is decreasing with qunidine.

Prophylaxis is best accomplished by general measures directed toward removing precipitating causes and treating underlying conditions e.g. radio-active iodine for thyrotoxicosis. The number and frequency of paroxysms may be further reduced wth 0.2 Gm. quinidine sulphate orally t. i. d.

References

1. Harrison, T. R.: Principles of Internal Medicine, 2nd Edition.
2. White, Paul: Heart Disease, 4th Edition.
3. Davidson, Sir Stanley: Principles and Practice of Medicine, 3rd Edition.
4. Levine, S.: Clinical Heart Disease, 4th Edition.
5. Prinzmetal: The Auricular Arrhythmias.

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*Corrections to page 182.

Table 3 <2 | 120

Children

(1) Colloids: 4 cc./10 lb. x % body sur-
(2) Electrolytes: 4 cc./10 lb. x % body

*With apologies to Dr. A. W. Farmer and Dr. D. C. Robertson.

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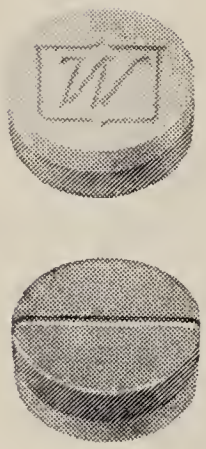

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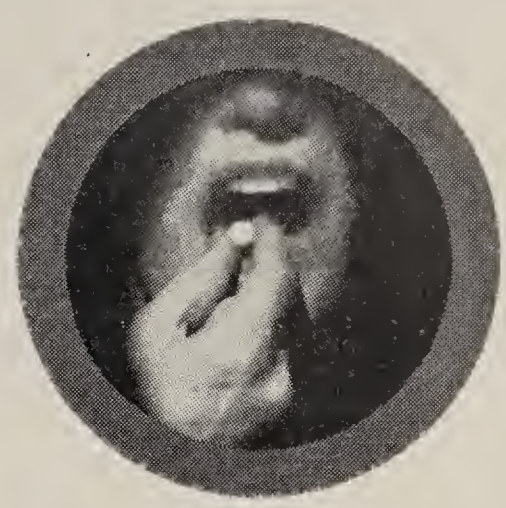

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*Abramson, D.: Personal communication.

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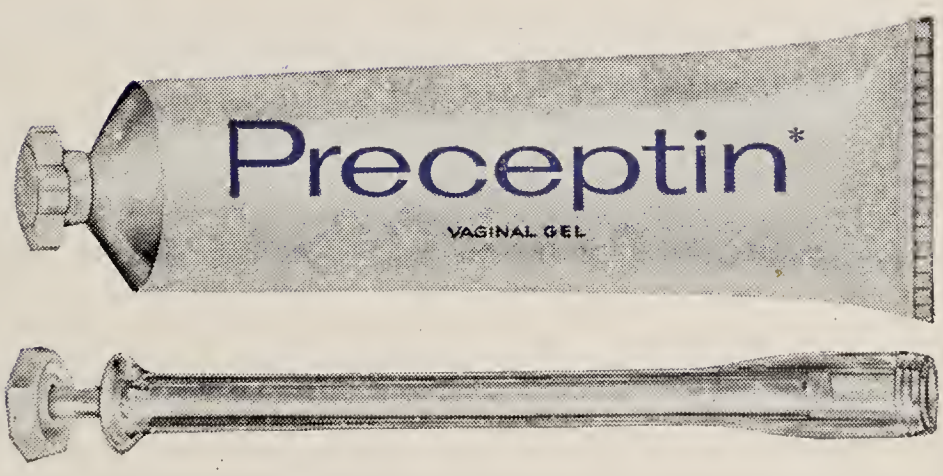

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